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Nutrigenomics in personalized metabolic care: Toward precision nutrition.

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Introduction

The global rise in metabolic disorders including obesity, type 2 diabetes, cardiovascular disease, and dyslipidemia has sparked a revolution in how we understand and treat nutrition-related illnesses. One of the most promising advances is nutrigenomics, a field that investigates how individual genetic variation affects responses to nutrients and how nutrients, in turn, influence gene expression. Traditional dietary guidelines follow a "one-size-fits-all" model, which often fails to produce consistent health outcomes. In contrast, precision nutrition, powered by nutrigenomics, offers a personalized approach based on an individual's genetic, epigenetic, and metabolic profiles. This shift holds the potential to optimize dietary strategies for disease prevention and improved metabolic health. This article explores the foundation of nutrigenomics, its applications in metabolic disorders, and the challenges and future directions of integrating genetic data into routine dietary care [1].

Nutrigenomics is distinct from but related to nutrigenetics. While nutrigenetics examines how genetic variations affect responses to nutrients (e.g., lactose intolerance), nutrigenomics studies how nutrients alter gene expression and cellular function. Nutrients act as ligands for transcription factors, modifying gene expression directly. Dietary components can influence epigenetic mechanisms such as DNA methylation, histone modification, and non-coding RNA expression. Metabolites derived from food (e.g., SCFAs from fiber) act as signaling molecules, modulating inflammation, insulin sensitivity, and lipid metabolism [2].

Genes such as FTO and MC4R are linked to increased appetite and weight gain .Personalized interventions targeting energy-dense foods and physical activity can be tailored based on these genotypes. Studies show that individuals with

certain FTO variants may respond better to highprotein, calorie-controlled diets. Genetic variants in TCF7L2 influence insulin secretion and risk for T2D.People with high-risk genotypes benefit more from low-glycemic index diets and early lifestyle interventions.Nutrigenomic profiling can also guide responses to metformin and other glucose-lowering agents.Variants in APOE, CETP, and LPL affect lipid metabolism and cardiovascular risk [3].

For instance, individuals with the APOE4 allele are more sensitive to dietary saturated fats, requiring more aggressive lipid-lowering diets. Diets rich in omega-3 fatty acids, polyphenols, and antioxidants can modulate inflammation-related genes like IL-6, TNF-α, and NF-kB. These changes are especially important for individuals predisposed to chronic inflammation and insulin resistance. Genotyping panels (e.g., 23andMe, Nutrigenomix) allow for affordable and quick assessment of nutrition-related gene variants. Transcriptomics, proteomics, and metabolomics provide deeper insight into how nutrients alter biological pathways [4].

Machine learning models integrate genetic and lifestyle data to predict optimal diets for weight loss, blood sugar control, and lipid balance. Nutritional outcomes are rarely dictated by single genes. Polygenic risk scores and genegene/environment interactions complicate predictions. Many commercial nutrigenomic tests lack scientific validation or produce conflicting dietary recommendations. Genetic data raises potential concerns about data security, discrimination, and misuse by insurers or employers. Precision nutrition services remain limited to urban or high-income populations, potentially widening health disparities [5].

Conclusion

Nutrigenomics represents a transformative step in metabolic care, providing tailored nutritional

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strategies based on individual genetic and metabolic profiles. By moving away from generic guidelines and embracing precision nutrition, healthcare providers can more effectively prevent and manage metabolic diseases. Despite current challenges such as incomplete scientific understanding, regulatory gaps, and accessibility issues the future of nutrigenomics is promising. As technologies mature and datasets expand, personalized dietary plans will likely become a central component of preventive medicine, enhancing both clinical outcomes and quality of life across populations.

References

1. Storr M, Allescher HD. Esophageal pharmacology and treatment of priApry motility disorders. Diseases of the Esophagus. 1999;12(4):241-57.

- 2. Sifrim D, Janssens J, Vantrappen G. Failing deglutitive inhibition in priApry esophageal motility disorders. Gastroenterol. 1994;106(4):875-82.
- 3. Teitelbaum EN, Dunst CM, Reavis KM, et al. Clinical outcomes five years after POEM for treatment of priApry esophageal motility disorders. Surg Endosc. 2018;32(1):421-7.
- 4. Mazari-Hiriart M, Lopez-Vidal Y, Castillo-Rojas G, et al. Helicobacter pylori and other enteric bacteria in freshwater environments in Mexico City. Arch Med Res. 2001;32(5):458-67.
- 5. McGowan CC, Cover TL, Blaser MJ. Helicobacter pylori and gastric acid: biological and therapeutic implications. Gastroenterol. 1996;110(3):926-38.